2000) (9,10). Of the two case-patients, one soldier did not leave NDjamena during his 3-month tour of duty, whereas the other had been in contact with livestock in a flooded area before onset of symptoms. Contamination may have occurred through infected animals or mosquitoes, although sheep living in the area did not show any sign of disease (i.e., spontaneous abortions, deaths). The two cases we describe were self-limiting; however, deaths from this illness have been reported in nonepidemic settings in Central African Republic (11). Our data emphasize that healthcare providers should systematically consider Rift Valley fever as a diagnosis for febrile syndromes in persons returning from Africa, even in nonepidemic settings (12).

Jean Paul Durand,*
Michèle Bouloy,†
Laurent Richecoeur,‡
Christophe Nicolas Peyrefitte,*
and Hugues Tolou*

*Tropical Medicine Institute of the French Army Medical Corps (IMTSSA), Marseille, France; †Institut Pasteur, Paris, France; and ‡3ème Régiment d'Infanterie de Marine (RIMa), Vannes Cedex, France

References

1. Daubney R, Hudson JR, Graham PC. Enzootic hepatitis of Rift Valley fever, an undescribed virus disease of sheep, cattle and man from East Africa. Journal of Pathology and Bacteriology 1931; 34: 545–79.
2. Maurice Y. Premières constatations sérologiques sur l'incidence de la maladie de Wesselsbron et de la Fièvre de la Vallée du Rift chez les ovins et les ruminants sauvages du Tchad et du Cameroun. Rev Elev Méd Vét Pays Trop Revue d'Elevage et de Médecine Vétérinaire des Pays Tropicaux 1967;20:395–405.
3. Meegan JM, Hoogstraal H, Moussa MI. An epizootic of Rift Valley fever in Egypt in 1977. Vet Rec 1979;105:124–5.
4. Jouan A, Le Guenno B, Digoutte JP, Philippe B, Riou O, Adam F. An RVF epidemic in southern Mauritania. Ann Inst Pasteur Virol 1988;139:307–8.
5. Nasher AAW, Shiban AK, Eriyani MA, Aly Bourgy A, Al Kohlani AH, Benbrake M, et al. Outbreak of Rift Valley fever, Yemen, August–October 2000. Wkly Epidemiol Rec 2000;75:392–5.
6. Arishi H, Ageel A, Abdu Rahman M, Al Hazmi A, Arishi AR, Ayoola B, et al. Outbreak of Rift Valley fever, Saudi Arabia, August–November 2000. MMWR Morb Mortal Wkly Rep 2000;49:982–5.
7. Sall AA, de Zanotto PM, Sene OK, Zeller HG, Digoutte JP, Thiongane Y, et al. Genetic reassortment of Rift Valley fever virus in nature. J Virol 1999;73:8196–200.
8. Sall AA, de Zanotto PM, Zeller HG, Digoutte JP, Thiongane Y, Bouloy M. Variability of the NSs protein among Rift Valley fever virus isolates. J Gen Virol 1997;78:2853–8.
9. Miller BR, Godsey MS, Crabtree MM, Savage HM, Al-Mazrao Y, Al-Jeffri M, et al. Isolation and genetic characterization of Rift Valley fever virus from Aedes vexans arabiensis, Kindom of Saudi Arabia. Emerg Infect Dis 2002;8:1492–4.
10. Shoemaker T, Boulianne C, Vincent MJ, Pezzanile L, Al-Qahtani MM, Al-Mazrou Y, et al. Genetic analysis of viruses associated with emergence of Rift Valley fever in Saudi Arabia and Yemen, 2000–01. Emerg Infect Dis 2002;8:1415–20.
11. Meunier DMY, Madelon MC, Lesbordes JL, Georges AJ. La fièvre de la Vallée du Rift et les phléboviroses en République Centrafricaine. Bull Soc Pathol Exot Filiales 1988;81:49–57.
12. Durand JP, Richecoeur L, Peyrefitte C, Boutin JP, Davoust B, Zeller H, et al. La Fièvre de la Vallée du Rift: infections sporadiques de militaires français hors des zones d'épidémies actuellement connues. Med Trop (Mars) 2002;62:291–4.

Address for correspondence: Jean Paul Durand, Laboratoire Associé au Centre National de Référence des Arbovirus, Unité de Virologie, IMTSSA, BP 46, 13998 Marseille Armées, France; fax: 01 40 61 31 51; email: imtssa.vro@wanadoo.fr

Corynebacterium ulcerans Diphtheria in Japan

To the Editor: *Corynebacterium ulcerans* causes a zoonotic infection similar to diphtheria, which is caused by *C. diphtheriae*. Studies indicate that signs and symptoms of a diphtheria-like illness caused by *C. ulcerans* are milder than those caused by *C. diphtheriae*. However, some strains of *C. ulcerans* produce potent diphtheria toxin and may cause severe symptoms similar to those caused by *C. diphtheriae* (1). We report a case of a diphtheria-like illness caused by *C. ulcerans* infection.

A previously healthy 52-year-old woman first noticed hoarseness approximately 3 days before admission to the hospital. On February 16, 2001, severe dyspnea and fever developed, and the patient was referred to the emergency room of the Asahi General Hospital by her private practitioner. Physical examination indicated a large stridor, which could be heard without using a stethoscope. Cyanosis was not observed. The endoscopic examination showed a thick white coat covering the nasopharynx and laryngeal vestibulum, and subglottic constriction was also observed. A chest x-ray showed diffuse infiltrates in both lungs. Pertinent laboratory findings on admission included leukocyte count of 16.8 x $10^3/\mu L$ and C-reactive protein of 20.0 mg/dL. The serum level of liver transaminase was normal, and both Wassermann reaction and anti-HIV antibody tests were negative. Pharyngolaryngitis and pneumonia was diagnosed in the patient. Because of severe dyspnea, intubation was performed, which caused sudden and unexpected exacerbation of the condition. Severe cyanosis subsequently developed. Extubation was immediately performed, and a thick white material was found to be filling the lumen of the endotracheal tube. Reintubation was performed, and dyspnea subsided. The patient was hospitalized in the intensive-care unit. Sulbactam sodium/ampicillin sodium (6 g per day) was intravenously administered for 4 days; however, the symptoms were not much improved. The symptoms were most consistent with those of diphtheria. Therefore, the patient was subsequently placed

on erythromycin (1.0 g/day) and quickly responded to this treatment without administration of diphtheria antiserum. Erythromycin was intravenously administered at 1 g per day for 9 days, then orally administered at 1,200 mg per day for the next 14 days. Throughout the hospitalization, no complication occurred, and no abnormalities were noted in the electrocardiograms or in the patient's neurologic status. The patient was discharged uneventfully, and no serious sequelae were noted for 20 months. History of immunization for diphtheria was not known.

After the hospitalization for this acute illness, a laboratory report showed that *C. ulcerans* was cultured from the thick white coat of the throat. No other bacteria were found. The National Institute of Infectious Diseases in Tokyo later confirmed identification of the bacteria. By using Elek's test, Vero cell toxicity, and polymerase chain reaction for toxigene, this strain of *C. ulcerans* was proven to produce diphtheria toxin identical to *C. diphtheriae* (2–4). Although administering appropriate antibiotics as well as antitoxin is a standard of care for patients with diphtheria, antitoxin was not given to this patient because of her rapid response to the erythromycin therapy.

C. ulcerans infections in humans occur after drinking unpasteurized milk or coming in contact with dairy animals or their waste (5,6). However, person-to-person transmission of *C. ulcerans* has not been reported, and in some cases, the route of transmission is not clear (7). Recently, *C. ulcerans*-producing diphtheria toxin was isolated in the United Kingdom from cats with nasal discharge (8).

Our patient did not have direct contact with dairy livestock or unpasteurized dairy products; however, more than 10 dairy farms are scattered around her home. Moreover, she kept nearly 20 cats in her house and had been scratched by a stray cat a week before illness developed. This stray cat, which had rhinorrhea and sneezing, had wandered into her house. The stray cat died before the patient became ill, and no further investigation could be made. Stray cats might well be one of the possible carriers of *C. ulcerans* and might have transmitted the bacteria to this patient. To our knowledge, a case of human infection caused by *C. ulcerans* has never been reported in Japan. On the basis of current experience, this bacterium does exist in Japan and can potentially cause a serious diphtheria-like illness in humans.

Akio Hatanaka,* Atsunobu Tsunoda,* Makoto Okamoto,* Kenji Ooe,* Akira Nakamura,* Masashi Miyakoshi,* Takako Komiya,† and Motohide Takahashi†

*Asahi General Hospital, Chiba, Japan; and †National Institute of Infectious Diseases, Tokyo, Japan

References

1. Kisely SR, Price S, Ward T. '*Corynebacterium ulcerans*': a potential cause of diphtheria. Commun Dis Rep CDR Rev 1994;4:R63–4.
2. Reinhardt DJ, Lee A, Popovic T. Antitoxin-in-membrane and antitoxin-in-well assays for detection of toxigenic *Corynebacterium diphtheriae*. J Clin Microbiol 1998;36: 207–10.
3. Nakao H, Popovic T. Development of a direct PCR assay for detection of the diphtheria toxin gene. J Clin Microbiol 1997;35:1651–5.
4. Miyamura K, Nishio S, Ito A, Murata R, Kono R. Micro cell culture method for detection of diphtheria toxin and antitoxin titers by VERO cells. J Biol Stand 1974;2:189–201.
5. Bostock AD, Gilbert FR, Lewis D, Smith DCM. *Corynebacterium ulcerans* infection associated with untreated milk. J Infect 1984;9:286–8.
6. Barret NJ. Communicable disease associated with milk and dairy products in England and Wales: 1983–84. J Infect 1986;12:265–72.
7. Pers C. Infection due to "*Corynebacterium ulcerans*," producing diphtheria toxin. Acta Pathol Microbiol Immunol Scand [B] 1987;95:361.
8. Taylor DJ, Efstratlou A, Reilly WJ. Diphtheria toxin production by *Corynebacterium ulcerans* from cats. Vet Rec 2002;150:355.

Address for correspondence: Atsunobu Tsunoda, Department of Otolaryngology, Asahi General Hospital, Asahi I-1326, Chiba, 289-2511, Japan; fax: 81-479-63-8580; email: atsunoda@mac.com

Salmonella in Birds Migrating through Sweden

To the Editor: To determine how common *Salmonella* infection is in the migrating wild bird population, we considered the biology of the bacterium and that of its avian hosts. Previous studies have attempted to determine in which stages wild birds become infected, how infections are acquired, and how this information should be translated into epidemiologic risk assessments for human and animal health. For instance, most published studies originate from small epizootics and are of either dead birds at feeding stations (1) or infected birds in or around barns where the livestock has *Salmonella* infection (2). This bias has important consequences, as the natural prevalence of *Salmonella* in the non-epizootic situation likely is overestimated. Finding infected birds close to a barn with infected cattle does not prove that transmission occurred from the birds to the animals. In addition, an epizootic at a feeding station does not prove that *Salmonella* normally occurs in the inflicted bird species, as the birds could have became infected through proximity to the infected animals, or in the case of the bird feeder, through feed contaminated from an unknown source. We need baseline surveillance data on the prevalence of *Salmonella* in non-epizootic situations, in healthy bird communities and in different